the dopaminergic system.[3] Thus the usual release of extrapyramidal overactivity by neuroleptics blocking dopamine receptors would be expected to occur less frequently in demented patients. In view of the considerable pressure, which is likely to increase in the future, on inadequate psychogeriatric services from an aging population the use of haloperidol should, I feel, be recommended as the drug of choice to help keep more elderly patients in the community. In 18 months, of 184 domiciliary visits, 40 were to demented patients (22%); this reflects the seriousness of this problem in general adult psychiatric practice in a representative urban community.

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[1] Oldham, A J, and Bott, M, *Acta Psychiatrica Scandinavica*, 1971, **47**, 369.
[2] Tobin, J M, *Geriatrics*, 1970, **25**, 119.
[3] Davies, P, and Maloney, A J F, *Lancet*, 1976, **2**, 1403.

Alcoholic liver disease

SIR,—I have read with great interest the paper on the changing pattern of alcoholic liver disease in Great Britain by Dr N Krasner and others (11 June, p 1497).

In the district of Italy (north-east) where I work the daily average per caput alcohol consumption is very high, alcoholic liver disease is widespread, and the proportion of cirrhosis attributed to excessive drinking is greater (close to 90%) than that reported in British surveys.[1]

Our experience with long-term survival rates of all patients with alcoholic liver disease in relation to continuing or stopping drinking agrees completely with the figures given by Dr Krasner and his colleagues. Indeed, alcoholic liver disease even in advanced cases seems to bear a fairly good prognosis (excluding early deaths from irreversible complications) provided that total abstinence can be achieved and indefinitely maintained.

Our findings differ from those of the authors, however, with regard to sex-related differences. In fact, while perhaps agreeing about a higher incidence of serious forms of alcoholic hepatitis in women, we do not see a significant sex-related difference in the long-term prognosis of alcoholic liver disease. Nor does our experience agree with the finding that the incidence of alcoholic hyaline on liver biopsy and of serum autoantibodies is significantly higher in women. The latter finding, indeed, is extremely uncommon in our patients (of both sexes) with alcoholic liver disease.

Although in non-alcoholic chronic hepatitis and in a few cases of alcoholic liver disease it seems undoubtedly to be so, the inference that "immune mechanisms may play a part in the pathogenesis and progression of alcoholic liver disease in women" should be accepted, in our experience, very cautiously.

Do our divergent findings reflect a genetic difference of our population or could they be related to the different type of alcoholic beverage (wine) traditionally consumed in this area?

While not quoting the serum levels of IgA, the authors report a predominant increase in the IgM and IgG classes in women with cirrhosis, a sex-related feature we are again unable to confirm. Our finding of a higher proportional increase of IgA in alcoholic liver disease is confirmed by others,[2] and in our experience it is so constant that we would suggest that it be regarded as an additional pointer to an alcoholic aetiology in hepatic disease.

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[1] Basile, A, *Difesa Sociale*, 1975, **3**, 287.
[2] Doniach, D, and Walker, G, in *Progress in Liver Disease*, eds H Popper, and F Schaffner, vol 4, p 381. New York, Grune and Stratton, 1972.

Abdominal tuberculosis in Britain

SIR,—We write to support your leading article (18 June, p 1557) in which you point out that one should not be complacent that abdominal tuberculosis is a rarity in the indigenous population of Britain. You refer to our observation[1] in which 14 out of 15 patients seen in a five-year period were immigrants. However, in recent months we have seen four patients with abdominal tuberculosis, all people of English extraction and background. One was a young woman with ileocolitis and active pulmonary tuberculosis, another a teenage girl with tuberculous peritonitis, and two were middle-aged persons with small-bowel tuberculous conditions.

Clearly it is important that all clinicians managing abdominal problems be aware of the possibility of tuberculosis, whether the patient is an immigrant or otherwise.

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[1] Mandal, B K, and Schofield, P F, *Practitioner*, 1976, **216**, 683.

Carbon dioxide-dependent Staphylococcus aureus from abscess

SIR,—I am reporting this case as I have not come across any other report of isolation of such a strain from clinical materials.

A specimen of pus was sent to our laboratory from an abscess in the neck of a 31-year-old woman. Gram staining of a direct smear showed plenty of pus cells but no organism. No acid-fast bacilli were seen on Ziehl-Neelsen staining. Culture plates examined next day showed a pure growth on the blood agar plate incubated anaerobically but no growth on the plate incubated aerobically. The colonies looked like those of staphylococci, though were smaller in size (because of anaerobic conditions). Gram staining and coagulase testing indicated that the organism was *Staphylococcus aureus*.

Because of the absence of growth in aerobic conditions further tests and subcultures were done. On subculture the organism failed to grow aerobically but showed good growth anaerobically. We use BBL Gas-pak for anaerobiosis. In addition to producing anaerobiosis, this also supplies carbon dioxide. To see the effect of carbon dioxide the next subcultures were done in three conditions: (*a*) anaerobically with carbon dioxide, (*b*) aerobically with carbon dioxide, and (*c*) aerobically without carbon dioxide. Growths were obtained in (*a*) and (*b*) but not in (*c*). Colonies on (*b*) were larger in size than those on (*a*). The same results were obtained on a series of subcultures done over several days. On further identification tests the strain was found to be coagulase positive, DNAse positive, phosphatase positive, fermentative on oxidation/fermentation reaction, and matched well into the Cowan and Steel table[1] for *Staph aureus*, except that it did not produce acid from mannitol. However, there are probably a few strains of *Staph aureus* which may not ferment mannitol,[2] and no Gram-positive cocci other than *Staph aureus* would give positive reactions on all of coagulase, DNAse, and phosphatase tests and grow anaerobically.[1 2]

Thus it was probably a carbon dioxide-dependent strain of *Staph aureus*, which would have been missed if we had not used Gas-pak for anaerobiosis. The organism was sensitive to all antibiotics except penicillin and tetracycline.

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[1] Cowan, S T, *Cowan and Steel's Manual for the Identification of Medical Bacteria*, 2nd edn. London, Cambridge University Press, 1974.
[3] Wilson, G S, and Miles, A A, *Topley and Wilson's Principles of Bacteriology, Virology and Immunology*, 6th edn. London, Arnold, 1975.

General practitioner prescribing costs

SIR,—In a recent leading article (12 March, p 670) you quite justifiably drew attention yet again to general practitioners' prescribing costs, at present about £15 000 worth of drugs a year being handed over by each practitioner.

It was especially shocking, therefore, to hear a colleague who has recently successfully completed a vocational training course in general practice, old MRCGP and all, state blithely that drug costs "did not influence at all" his choice of medication. Further inquiry revealed that no formal instruction, nor even general guidance, in keeping prescribing costs down had been given during the three-year course. What a glorious missed opportunity for producing real economies in an overstretched NHS budget!

I wonder if this is the universal UK experience or if somewhere the GPs of the future *are* taught about expensive drugs and the need to use their cheaper equivalents?

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Car allowance for consultants

SIR,—Among the consultants' many grievances, one causing most resentment at present is the question of car allowances. This is particularly so among full-timers, myself included, as it has just cost me over £2000 of tax-paid income to change my modest 5½-year-old Austin 1800 for an equally modest Ford Cortina, for which, of course, I will receive no allowance whatsoever.

At the same time I note that general practitioner trainees have had *their* car allowance raised to £1125 per annum "in accordance with the agreed formula for linking the trainee car allowance to the AA's schedule of motoring costs."

If it is right for them why not for us?

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